Some Issues in the Epidemiology of Schizophrenia
Author(s): August B. Hollingshead
Source: *American Sociological Review*, Vol. 26, No. 1 (Feb., 1961), pp. 5-13
Published by: American Sociological Association
Stable URL: http://www.jstor.org/stable/2090507
Accessed: 09-06-2016 17:18 UTC

REFERENCES
Linked references are available on JSTOR for this article:
http://www.jstor.org/stable/2090507?seq=1&cid=pdf-reference#references_tab_contents
You may need to log in to JSTOR to access the linked references.

Your use of the JSTOR archive indicates your acceptance of the Terms & Conditions of Use, available at
http://about.jstor.org/terms

JSTOR is a not-for-profit service that helps scholars, researchers, and students discover, use, and build upon a wide range of content in a trusted digital archive. We use information technology and tools to increase productivity and facilitate new forms of scholarship. For more information about JSTOR, please contact support@jstor.org.

Sage Publications, Inc., American Sociological Association are collaborating with JSTOR to digitize, preserve and extend access to *American Sociological Review*

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms

AMERICAN SOCIOLOGICAL REVIEW

February 1961 Volume 26, Number 1

SOME ISSUES IN THE EPIDEMIOLOGY OF SCHIZOPHRENIA *

AUGUST B. HOLLINGSHEAD
Yale University

Clinical, laboratory and epidemiological methods help man understand health and disease. Epidemiology seeks to determine who develops a disease, when, and under what conditions. Schizophrenia is a disabling illness of unknown etiology. Epidemiology may be the appropriate method to help man unravel the causative factors which give rise to it. Three issues in an epidemiological study of schizophrenia are discussed: the validity and reliability of psychiatric diagnoses, the nature of the population to be studied, and the possible ways sociologists can contribute to the exploration of interrelations between socio-cultural factors and the presence or absence of schizophrenia in a population.

THIS lecture may be viewed as prolegomena to a field study of schizophrenia. I have selected schizophrenia as the focus of discussion for several reasons. Schizophrenia disables; it does not kill. Approximately one hospital bed out of four in the United States is occupied by a schizophrenic patient, who has been hospitalized—on the average —for several years. The schizophrenic is always with us. How, and why, he comes into existence is an enigma. When society is faced with an unsolved health problem as severe as schizophrenia, research is not only worthwhile, but necessary. The strategic objective is to gain the knowledge to control the problem. The requisite knowledge may not be forthcoming in this generation, but we should proceed to the task imbued with the conviction that the rigorous application of scientific methods will give us the knowledge we need to solve the problem at hand.

EPIDEMIOLOGY

Medicine, as art and science, has developed three ways to help man understand health and disease. They are the clinical, laboratory, and epidemiological methods. Categorized briefly, the *clinical* method is directed toward individuals suffering from disease. The clinician focuses his attention upon the patient before him. The questions he asks, and the techniques he uses during the examination establish, in his judgment, the health or illness of the patient. To the clinician "health" is the absence of disease; "sickness" is the presence of signs and symptoms of disease. The *laboratory* method examines fluids, excretions, or tissues taken from the bodies of patients. The laboratory scientist may not see the patient. He directs his attention to the disease process reflected in the fluid or tissue he examines. The *epidemiological* method studies disease and phenomena associated with disease in a population of individuals; some are "sick," others are "well."

Epidemiology is not the study of epidemics, even though this is a common belief. The Greek phrase *"epi-demos"*—concerning the people—should be of as much interest to sociologists as to physicians. The focus of epidemiology is to relate the ways of living of a population, or a specific sub-group in it, to the observed distribution of a partic-

* The MacIver Lecture presented before the Pacific Sociological Association, Spokane, Washington, April, 1960.

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms

ular disease.[1] The epidemiologist tries to determine who develops the disease, when, and under what conditions.[2]

Goldberger's [3] researches on pellagra are a classic illustration of epidemiological method. In the early years of this century, pellagra was a common disease in the southern part of the United States. It was believed to be spread from person to person by infection. Goldberger first studied pellagra in a mental hospital. He saw that some patients suffered from pellagra when they were admitted, whereas others developed the disease in the hospital. Goldberger observed close contacts between members of the staff and the patients, but no staff members suffered from pellagra. The patients and staff were in intimate contact on the wards day after day; the patients developed pellagra, but the staff did not. Goldberger inferred from this fact that pellagra is not an infectious disease. He then looked for something that differentiated the two groups. He noted that the staff did not eat with the patients. Furthermore, the staff ate different foods. He developed the working hypothesis that differences in diet might explain why patients developed pellagra, but the staff did not. Goldberger then worked with a colleague, who tried an improved diet on a series of women patients afflicted with pellagra. The women responded remarkably to this diet. Other institutions where pellagra was common were then studied. In one orphanage, for example, pellagra was seen in children above six years of age. Research showed that children under six years of age were given two cups of fresh milk daily; children six years of age and older were not given any milk, and fresh meat only once a week. Again the experimental method was used to test the hypothesis that diet is related to the development of pellagra. All the children in the orphanage were given milk every day, and fresh meat four times a week for a year. No case of pellagra developed in this orphanage during the year of the experiment.

In another experiment, a group of mental patients were given special diets for a year: no one developed the disease, whereas forty-seven per cent of the patients in a control group, in the same hospital, had a recurrence of pellagra. Other studies included the experimental development of pellagra in convicts. Diet was the answer; but what ingredients in the diet? Goldberger, and those who have followed him, traced the etiology of pellagra to the vitamin B complex, nicotinic acid, and the action of tryptophane.

This sketch indicates that the epidemiological method is inductive and inferential. The co-existence of attributes, as well as variables, with health, or disease, is determined by studying a defined population and its constituent parts in time, place, and circumstance. Attributes and variables pertinent to epidemiological study include such things as sex, race, age, occupation, education, diet, family composition, group customs, and personal habits. In addition, it encompasses genetic composition, biochemistry, other forms of plant and animal life, and geographical environment. In brief, epidemiology is the application of ecological principles to the study of health, as well as disease, in a society. Its working hypotheses are conditional: If *this,* then *that.*

The clinical, laboratory, and epidemiological methods have a common interest: disease in individuals. The clinician, the laboratory analyst, and the epidemiologist utilize the findings of his colleagues in adjacent fields to solve problems posed by disease. The triumph of the epidemiological method has been its utility in helping researchers trace out, step by step, interdependencies between the life ways of individuals and the appearance or non-appearance of disease in a population. The epidemiological method should be useful in our efforts to unravel the etiology of the tangled skein of human misery the medical profession refers to as schizophrenia.

SCHIZOPHRENIA

Schizophrenia has been recognized as a disease for a century. In 1860 a case of a

[1] J. N. Morris, "Uses of Epidemiology," *British Medical Journal,* (August 13, 1955), pp. 395–410.

[2] Ernest M. Gruenberg, "The Epidemiology of Mental Disease," *Scientific American,* 190 (January, 1954), pp. 38–42.

[3] J. Goldberger, *Public Health Reports,* Washington, D. C., 1914, Volume XXIX, p. 1683; J. Goldberger, G. A. Wheeler, and E. Syden-stricker, *Public Health Reports,* Washington, D. C., 1920, Volume XXXV, p. 648; W. F. Lorenz, *Public Health Reports,* Washington, D. C., 1914, Volume XXIX, p. 2357.

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms

young male, who had "degenerated into a state of dementia," was described.[4] Three years later variants of this psychosis [5] were identified. By the end of the nineteenth century *dementia praecox* had been incorporated into Kraepelin's nosological scheme.[6] The name "schizophrenia" was introduced into the literature in 1911.[7] The term "schizophrenia" has replaced almost entirely the earlier *"dementia praecox."*

Six types of theories have been put forward since 1900 to account for the etiology of schizophrenia. The theory of a pathological lesion in the brain was enunciated first in 1904.[8] After Noguchi,[9] in 1911, demonstrated that general paresis is traceable to an infection of *Treponema pallidum,* the "schizococcus" was pursued with vigor,[10] but without success.[11] The search for an infectious agent in schizophrenia goes on, but the uncritical enthusiasm of earlier days is lacking.

The second type of etiological theory is biochemical in origin. Biochemical explanations of mental disorders are as old as Hippocrates. He believed changes in the blood and the humors of the brain accounted for certain psychological aberrations. However, it was Thudicum, the father of modern neuropsychiatry, who made the classic statement of this doctrine. Thudicum wrote:

> "Many forms of insanity are unquestionably the external manifestations of the effects upon the brain of substances of poisons fermented within the body, just as mental aberrations accompanying chronic alcoholic intoxication are the accumulated effects of a relatively simple poison fermented out of the body.[12]

Kraepelin accepted Thudicum's doctrine. He believed that schizophrenia was caused by faulty metabolism, but he was never able to prove it. A recent review of biochemical research on schizophrenia concludes that none of this research demonstrates a biochemical etiology.[13]

The third type of theory assumes a genetic etiology. Advances in biochemical techniques have led to renewed interest in the long-standing idea that schizophrenia is traceable to faulty heredity. Kallmann insists that schizophrenia is transmitted from parents to children through a single recessive gene.[14] Other scholars working in the new and specialized area of genetic epidemiology are not as confident.[15] This theoretical approach has not been explored adequately; its validity remains in doubt.

The fourth type of theory, the psychogenic approach, is traceable to Freud. However, it was his student and colleague, Jung,

---

[4] B. Morel, *Traités des maladies mentales,* Paris: Masson, 1860, p. 566.

[5] J. L. Kahlbaum, *Gruppierung der psychischen Krankheiten,* Danzig: Kafemann, 1863.

[6] E. Kraepelin, *Dementia Praecox and Paraphrenia,* translated from the Eighth German Edition, Edinburgh: Livingston, 1925.

[7] E. Bleuler, *Dementia Praecox, oder die Gruppe der Schizophrenien,* 1911, translated into English by Joseph Tinken, New York: International Universities Press, 1950; p. 1.

[8] A. Alzheimer, "Histologische Studien zur Differnzialdiagnose der Progressiven Paralyse," *Histologische und Histopathologische Arbeiten ueber die Grosshirnrinde mit besonderer Beruecksichtingung der Pathologischen Anatomie der Geisteskrankheiten,* Jena: Gustav Fischer, 1904, Volume I, p. 18; F. Nissl, *Zur Histopathologie der Paralytischen Rindenerkrankung,* Jena: Gustav Fischer, 1904, Volume I, p. 315.

[9] H. Noguchi and J. W. Moore, "A Demonstration of *Treponema Pallidum* in the Brain in Cases of General Paralysis," *Journal of Experimental Medicine,* 17 (1913), p. 232.

[10] Walter L. Bruetsch, "Mental Disorders Arising from Organic Diseases," in *The Biology of Mental Health and Disease* (108 authors), New York: Hoeber, 1952, pp. 303–326.

[11] Henry Cotton's ill-starred "Focal Infection Theory" may be traced to this school of thought. For a discussion of the point, see: Joseph Zubin, "Biometric Methods in Psychopathology," in Paul H. Hoch and Joseph Zubin, editors, *Depression,* New York: Grune and Stratton, 1954, p. 126.

[12] J. W. L. Thudicum, *A Treatise on the Chemical Constitution of the Brain,* London: Balliere, Tindall, and Cox, 1884.

[13] Seymour S. Kety, "Biochemical Theories of Schizophrenia," Part I, *Science,* 129 (May 22, 1959), pp. 1528–1532; Seymour S. Kety, "Biochemical Theories of Schizophrenia," Part II, *Science,* 129 (June 12, 1959), pp. 1590–1596.

[14] Franz J. Kallmann, "Genetic Aspects of Psychoses," in *The Biology of Mental Health and Disease, op. cit.,* pp. 283–303, especially p. 290; Franz J. Kallmann, "The Genetics of Mental Illness," in Silvano Arieti, editor, *American Handbook of Psychiatry,* New York: Basic Books, 1959, Volume I, pp. 175–196.

[15] Jan A. Böök, "Genetics of Schizophrenia," in *Work Conference in the Mental Disorders,* New York City, (February 15–19, 1959), pp. 300–301 and 538–542, (mimeographed); Seymour S. Kety, *op. cit.,* p. 1594.

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms

who first applied the theoretical structure of psychoanalytic thought to the etiology of schizophrenia.[16] The psychoanalytic position has been restated in numerous ways by more recent writers. Its validity still rests upon faith and assertion.

The fifth type of etiological theory is a sociogenic one. It developed out of psychogenic theory. Sullivan's [17] theory of disordered relations between a child and his parents represented a deviation, if not a clean break, from the Freudian position. The most recent variant of the interpersonal explanation for schizophrenia is the "double bind" hypothesis enunciated by Bateson and his associates.[18] The "double bind" theory is based on the proposition that schizophrenia is the product of faulty communication between human beings. An individual who is enmeshed in social situations, where he receives conflicting messages on different psychological levels, develops deviant response patterns to handle the messages he receives. The theory assumes that schizophrenia is the individual's adjustment to a pathogenic family environment—a family in which an individual "can't win" by acting in ways our society calls "normal."

The sixth variety of etiological theory is eclectic. Adolf Meyer accepted from Kraepelin and Bleuler the concept that schizophrenia is a nosological entity, but he rejected their ideas of etiology.[19] Meyer taught that schizophrenia is a reaction to the forces which impinge upon the schizophrenic during the course of his life. Meyer insisted that schizophrenia, and other functional mental illnesses, have multiple etiological bases. Every individual is a product of many forces and experiences—heredity, body chemistry, nervous system, family, station in life, psychological experiences, and so on. The psychobiological approach is represented currently by Leighton.[20]

The different etiological theories of schizophrenia have been substantiated only to the satisfaction of their protagonists. As a consequence, controversies revolve around the nature, phenomenology, onset, and course of this illness. What we know of schizophrenia has been derived almost entirely from the observation of hospitalized cases. The classic psychiatric approach has been to reconstruct, *post factum,* in elaborate detail, a case history, or a few case histories. Much of this writing has been *ad hoc* speculation to illustrate the theoretical position of the author. When an investigator looks hard enough for an explanation of something he sees, or thinks he sees, he usually finds it. In the process he may create a myth, mistake it for reality, then vehemently defend his position. Meanwhile, the search for knowledge is diverted.

Current controversies over schizophrenia are a heritage, in large part, of nineteenth century theories of disease. Throughout the nineteenth century, formal medical theory taught that the etiology of all diseases could be traced to constitutional predisposition, to changes in tissue, or to changes in cellular structure and function. Disease was dysfunction within the physiological system, however caused. The view was compatible with the Morgagnian theory of pathology. Giovanni Morgagni (1682–1771), the eighteenth century founder of modern pathology, taught that every symptom of disease reflects an underlying *lesion* in the body. Morgagni stressed pathology which could be seen, felt, weighed, or identified by microscopic, histological, or chemical analyses. The researches

---

[16] C. G. Jung, *The Psychology of Dementia Praecox,* Nervous and Mental Disease Monograph Number 30, New York: Nervous and Mental Disease Publishing Company, 1936. This book was written in 1903; this was nine years before Jung had his now well-known rift with Freud.

[17] Harry S. Sullivan, *Clinical Studies in Psychiatry,* New York: Norton, 1956; Harry S. Sullivan, *Conceptions of Modern Psychiatry,* Washington, D. C.: W. A. White Psychiatric Association, 1946; Harry S. Sullivan, *The Interpersonal Theory of Psychiatry,* New York: Norton, 1953.

[18] Gregory Bateson, Don D. Jackson, Jay Haley, and John Weakland, "Toward a Theory of Schizophrenia," *Behavioral Science,* 1 (October, 1956), pp. 251–264; Jay Haley, "An Interactional Description of Schizophrenia," *Psychiatry: Journal for the Study of Interpersonal Processes,* 22 (November, 1959), pp. 321–332; Jay Haley, "The Family of the Schizophrenic: A Model System," *The Journal of Nervous and Mental Disease,* 129 (October, 1959), pp. 357–374.

[19] Adolf Meyer, "Fundamental Conceptions of Dementia Praecox," *British Medical Journal,* 1906, reprinted in A. Lief, *The Commonsense Psychiatry of Dr. Adolf Meyer,* New York: McGraw-Hill, 1958, pp. 247–259.

[20] Alexander Leighton, *My Name Is Legion,* New York: Basic Books, 1959.

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms

of Louis Pasteur (1822–1895), Robert Koch (1843–1910), and other nineteenth century scientists strengthened the doctrine that a specific disease is caused by a particular pathogenic organism. In the early years of the twentieth century, every discovery in microbiology and organic chemistry intensified the search for the specific agents which cause disease. When the researches of Gregor Mendel (1822–1884) became known, in the early years of the present century, the traditional medical assumption of constitutional predisposition in disease was bulwarked by genetic evidence.

Schizophrenia is of so much interest to psychiatrists that the Second International Congress for Psychiatry, held in 1957, limited its program to this illness. In spite of this interest—perhaps because of it—there was not a single concept or piece of evidence put forward by a participant that was not rejected or challenged by someone else. Even the name "schizophrenia" was disapproved of by several people.[21] Psychiatrists, in brief, disagree about the phenomenology, etiology, and therapeutic course of schizophrenia. Moreover, there is controversy over what points they agree or disagree. This is confusing to a non-psychiatrist.

The fact is, we do not know what causes the disease, disorder, or reaction the psychiatrist labels schizophrenia. Epidemiological study may furnish the answer. I do not say it will. At the very least, it will provide a different perspective on the problem. When we observe persons in their homes, their family and friendship groupings, their neighborhoods, at work and at play, we see them in their natural habitats. When we study persons and groups in the socio-cultural environments which enmesh them, we gain new knowledge about their motivations, aspirations, frustrations, conflicts, joys, and sorrows. Systematic and sophisticated study of the ecology of human life may unravel the etiological threads that lead to schizophrenia. I believe a series of carefully designed field studies of mental illnesses should be planned and carried out in future years. At least one aspect of the suggested studies might well be focused on the etiology of schizophrenia. When this is done, we shall face immediately three issues: (1) who shall be studied; (2) how reliable and valid are psychiatric diagnoses of functional mental illnesses; and (3) how can we relate socio-cultural factors to the presence or absence of schizophrenia?

ISSUES

(1) The Study Group. The first issue revolves around the population to be studied. Ideally, the entire population of a society should be included in a definitive study. However, we are not likely to achieve this desideratum for a long time. The permissible alternative is a probability sample of sufficient size to keep the standard error small. If we study a sample, other problems that are associated with schizophrenia, such as the age of onset, need to be considered. For example, should persons above sixty years of age be included? Should an *Index* individual or an entire family be studied? How far should family relationships be followed? Numerous additional questions should be considered in the planning phase of the suggested study.

Every experienced field researcher knows that before a study can succeed he must gain the cooperation of the persons in his sample. In a study dealing with a subject carrying the emotional charge that mental disease[22] does, gaining the support of a sample of individuals for the length of time essential to the kinds of studies envisaged here is likely to be a complicated, time-consuming, and exhausting task. Nevertheless, from the experience we have gained in an intensive field study of schizophrenia in Puerto Rico, I am convinced that the cooperation of adults in a sample can be gained. Our Puerto Rican study requires that each person drawn in the sample be given a two-and-one-half to three-hour psychiatric examination. Whether the examining psychiatrist diagnoses the sample person as schizophrenic or as having "no mental illness," we study him in elaborate detail. The schizophrenic person, as well as

[21] Silvano Arieti, "Schizophrenia: The Manifest Symptomatology, the Psychodynamic and Formal Mechanisms," in *American Handbook of Psychiatry, op. cit.*, p. 455.

[22] Elaine Cumming and John Cumming, *Closed Ranks,* Cambridge: Harvard University Press, 1957. This is an experiment in this area.

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms

one with "no mental illness"—and other adult members of their households—is interviewed by psychiatric social workers and social scientists in the home, from one-hundred-and-ten to one-hundred-and-twenty-five hours. We mention this to indicate a field study need not be overwhelmed by the difficulties of working with a population of non-patients, who are "sick" or "well."

Field studies of mental illnesses should not be "one shot" operations. The population should be studied for at least one year; but five, ten, or possibly twenty years would be more desirable. However, the longer the period of observation the more difficult the task of following individuals from place to place. Staff problems, too, become complicated in long-range studies. Nevertheless, in my judgment, such studies are necessary.

(2) Diagnosis. Diagnosis is a field problem of primary importance. Diagnosis is not easy even in diseases where there is an identifiable organic condition, and the clinician or researcher has a diagnostic instrument to aid him—such as an X-ray machine or a chemical test. The problem is more acute in schizophrenia. Here there is no chemical or physical test. Diagnosis is dependent solely upon the clinician's judgment. A number of different subjective factors may enter the process: the psychiatrist's conception of schizophrenia, the etiological doctrine he holds, how the person being examined thinks, talks, gestures, and the affect he presents. However, the subject's reactions may be responses to his cultural experiences and his judgments about the psychiatrist. Other factors—such as age, sex, race, religion, and social status—may enter into the diagnostic formulation.

Psychiatrists are concerned about the *reliability* and the *validity* of diagnoses. The reports of competent and responsible men who have wrestled with these questions show that the subject deserves attention. For example, Hoch recently reported that the ratio of first admissions diagnosed as manic depressives in comparison with schizophrenics reversed itself over a period of five years in one state hospital system. Hoch attributes this reversal not to a shift in the distribution of disease in the state's population, but to a change in personnel and policy in the hospital system.[23] Pasamanick supplemented this report with a statement of findings from the University hospital with which he is identified. Pasamanick found significant differences between the diagnostic classifications placed upon patients with functional psychoses from one ward to another. On one ward, the diagnoses changed when the ward administrator changed. The interesting thing is that the diagnoses had been made by residents, as well as the ward administrator, who was a trained psychiatrist.[24] The point to remember about these reports is that they are based upon hospitalized patients.

The problem of diagnosis is more complicated when a researcher is attempting to assess the mental status of a non-patient group. Leighton has furnished us some data on what happens when this is done. In the Mid-Town Study, New York City, six psychiatrists were asked to read the field protocols on fifty white, adult males. The psychiatrists were instructed to assess whether each man was mentally "well" or mentally "ill"; fifteen were placed in an equivocal category, and five individuals were diagnosed as "well." However, the five men who were diagnosed as "well" differed for each of the six psychiatrists. One psychiatrist's five "wells" were in another psychiatrist's "sickest" group.[25] Subsequent to this experience, the director of the project sat down with his fellow psychiatrists and worked out an operational procedure that produced *reliability* between the psychiatrists. But we do not know if the diagnoses they made are *valid,* or merely the product of consensus within the group. These reports indicate that the validity and the reliability of diagnosis in functional mental disorders represent difficult problems.

Currently, psychiatry does not have a standard test which researchers may use to diagnose any of the functional mental illnesses. A standardized, valid, diagnostic test would enable a researcher to determine the presence or absence of functional mental illness in individuals. Until this problem is solved, research into mental illness will con-

---

[23] Paul H. Hoch, *Work Conference in the Mental Disorders, op. cit.,* pp. 145–146.

[24] Benjamin Pasamanick, *Ibid.,* pp. 143–145.

[25] Alexander H. Leighton, *Ibid.,* pp. 147–148.

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms

tinue to be hampered. In sum, before we can move forward with confidence in epidemiological studies we need to establish criteria to differentiate a schizophrenic from a non-schizophrenic person. For present purposes, we shall assume this issue can be solved by careful planning, operational definitions, training of a field staff, and pilot studies.

(3) The Measurement of Risk. The third issue revolves around the question of how we may relate socio-cultural factors to the development of schizophrenia. This issue is the core of the epidemiological method. Epidemiological studies directed toward uncovering the etiology of a disease often pass through two principal phases: the descriptive and the determinative. The descriptive phase is concerned with relating the "sick" to the "well." The determinative phase tests inferences as to cause, developed in the descriptive phase. The essence of descriptive epidemiology is the comparison of risks in a population. During the descriptive phase of a study the population should be observed so carefully that a statement of the risk of developing the disease under examination is possible.

The measurement of comparative risks is stated as a rate. A rate is computed by dividing the number of cases of the disease by the population at risk.[26] The principal measures of risk are expressed in the concepts of incidence and prevalence. *Incidence* is a measure of *the way a disease attacks* a population, whereas *prevalence* is a measure of *the extent of suffering* in the population. Incidence is defined as the number of cases of a disease that develop in a population during a stated interval of time. Prevalence is the number of cases in a population at some point in time. Prevalence includes all cases active in a population, whether new or old, whereas only new cases are encompassed in incidence. Either incidence or prevalence has meaning only when it is viewed as a measure of the risks individuals in a defined population encounter of becoming ill of a specified disease, or suffering from it, at some point in time.[27]

Determination of the number of "active" cases in a defined population during an interval of time is a requisite step toward a solution of the problem of etiology. This means individuals who are "sick," but undiagnosed and not being treated, as well as persons who are under medical care. So far as mental illness is concerned, we cannot continue to base studies on treated cases only, and then attempt to draw inferences from this segment of the population of ill persons, as if it represented the universe of "sick" individuals. Who reaches a psychiatric facility may be more a product of the social system than of the disease process. Evidence is mounting that only a *portion* of individuals suffering from schizophrenia, or any other mental illness, are treated by psychiatric hospitals, clinics, and practitioners. The others are cared for by their families, non-psychiatric physicians, ministers, druggists, friends and neighbors, or through their own efforts.

[26] The procedure of stating the relationship of the "sick" to the "well" in the population at risk as a rate has at least four advantages:

First, it enables an investigator to see how many cases would be contributed by the population, or a sub-group, if the proportion of "sick" persons, or sub-groups, in the population were applied to a population of some standard size. By stating findings in terms of a rate per "standard unit" the researcher is able to make comparisons of the number of "cases" in population sub-groups of different sizes.

Second, by stating the relationship of the "sick" to the "well," as a ratio, the researcher may *adjust* the rate or ratio by holding one or more factors constant statistically. In this way, factors that may be confounding a calculated rate are controlled.

Third, rates may be stated as specific for sex, age, race, socio-economic status, religion, or any other defined criterion.

Fourth, rates may be tested for significance by standard statistical procedures.

[27] When a population is studied during a specified interval of time, each case of illness which is identified needs to be placed in one of four categories. These categories are:

First, cases which existed prior to the beginning of the interval of observation, which continue throughout the interval and still exist at the end of the interval.

Second, those which existed prior to the beginning of the interval and terminated during the interval.

Third, those which arise during the interval and still exist at the end of the interval.

Fourth, those which arise during the interval and terminate before the end of the interval.

These categories take into consideration the dynamic factors of additions to, and subtractions from, the number of cases of a specified disease. When these categories are combined, one can calculate the number of persons identifiable as suffering from a disease in a population during an interval of observation.

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms

Each individual in a field study focused on etiology ought to be examined at the beginning of a period of observation and again at the end of it. Two examinations are a minimum in a one-year study. Over a longer period, periodic examinations are indicated. An examination at the beginning and the end of the period of observation would provide the elementary data essential for the calculation of "true" prevalence and incidence rates. (I am making the assumption that a psychiatric examination can determine a "new" case of schizophrenia from an "old" one. This assumption itself deserves extended discusion.) Each rate ought to be determined with specific factors controlled statistically —such as age, sex, and socio-economic status. This procedure would enable the researcher to see if the amount of illness clusters in selected segments of the population.

The cases of schizophrenia that become active during an interval of observation would be of primary importance from the viewpoint of unraveling etiology. The "new" cases enable the researcher to determine how the disorder attacks individuals. The data should be controlled for specified attributes and variables, which the researcher suspects are related to the presence or absence of the disease. If the "new" cases cluster in a particular portion of the population, the researcher has located a possible clue to etiological factors. When this occurs the researcher understandably is encouraged. This is where he should exercise great caution. In his enthusiasm he may misinterpret clues and draw false inferences. A false lead will only confound future research. Observed differences may be a product of the research design, or the investigator may be drawing conclusions from incomparable data.[28]

When the comparison of rates reveals a clustering of illness in a particular portion of the population and a relative absence of the disease in other portions, the researcher is in a position to formulate working hypotheses regarding etiology. If coexistence is found between the presence or absence of disease and the way of life of an individual or social group, then the researcher should examine painstakingly the conditions—ecological, socio-cultural, and so on—under which individuals live who are high risks, as well as low risks. By close, systematic study of the life ways of individuals and groups the epidemiologist may be able to learn how the disease process is related to the circumstances in which it develops. Close scrutiny of the habits and customs of persons who are afflicted with the disease being studied, as well as a comparable series of individuals who are free of the disease, is a variant of the "treated" versus "control" procedures so widely used in experimental studies. The epidemiologist simulates experimental procedure by the comparison of two groups of differential risks.

Systematic field study of the processes which give rise to schizophrenic, as well as non-schizophrenic, behavior is indicated at the present time. When this is done, and I think it will be done eventually, socio-cultural factors should be studied along with heredity, biochemical processes, organic in-

[28] The need for caution is illustrated by the following incident: The biochemical laboratory of the National Institute of Mental Health was studying the urine of a sample of carefully diagnosed schizophrenic patients and a matched sample of non-schizophrenic individuals. The research was directed toward the determination of the quantity of phenolic acids excreted in a standardized interval of time. The researchers found four compounds in the urine from the schizophrenics that were present in significantly higher amounts than the same compounds in the urine of the control group. They also found one compound in the urine of the controls that was present in significantly higher amounts than in the schizophrenics. The researchers thought they had a real clue until a colleague in the laboratory, who had had experience as an agricultural chemist, pointed out that the four compounds found in the urine of the schizophrenics were metabolites of substances in coffee. The compound found in the urine of the normal control group was a derivative of the oil of wintergreen. The researchers then observed the activities of the schizophrenics, who were hospitalized, and questioned the normal controls, who were not hospitalized. They soon discovered that the schizophrenics drank several more cups of coffee each day than the normals. Moreover, the normals cleaned their teeth with commercial toothpastes; the schizophrenics did not. Oil of wintergreen is a common flavoring ingredient in toothpaste. The chemical findings for the schizophrenics, as well as the controls, may be attributed to the social situations in which the two groups lived. The source of this experience is: Seymour S. Kety, "Current Biochemical Theories of Schizophrenia," Lecture presented by The Yale Medical Society, New Haven, Connecticut: Yale University, November 10, 1959.

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms

sults, and psychological mechanisms. The possible etiological effects of each of these factors probably can be understood only in their concomitant interdependencies.

From the viewpoint of sociological theory, it would be desirable to postulate factors in the socio-cultural matrix which, directly or indirectly, give rise to disordered behavior in an individual. In doing this, the theorist should state clearly what he believes the connection is between socio-cultural factors, which impinge upon persons under defined conditions, and specified behavioral consequences. To investigate possible etiological linkages between socio-cultural variables and the development of schizophrenic behavior in the individual, the investigator needs to differentiate between independent and dependent variables. This elementary principle is overlooked commonly in psychiatric writing and research. In this field the tendency is to deal with many variables, on several levels, without specification.

SUMMARY AND CONCLUSION

To summarize this discussion:

First, epidemiological research directed toward the discovery of the etiology of schizophrenia is indicated at the present time. This research should be a study of this illness in populations, not merely in hospitals and clinics.

Second, no generally accepted etiological theory of schizophrenia is available for test or verification. This statement is made in full cognizance of the contributions that have been made to our understanding by psychogeneticists, as well as the recent attempts of social scientists to formulate theories which bridge the gap between the individual as an organism and the socio-cultural matrix that surrounds him. In sum, the necessary and sufficient causes of schizophrenia are matters of speculation.

Third, we should look forward to the day when we are able to build theoretical models which will forecast with precision:

A. The conditions which invariably give rise to the development of schizophrenia during the life of an individual if he lives, let us say, to the age of sixty. The suggested model should be phrased so that a researcher would be able to establish the validity of an etiological sequence so clearly that alternative explanations are untenable.

B. The conditions and processes in the lives of individuals which result in the appearance of other types of mental illnesses.

C. The conditions that are likely to result in "no mental illness" in individuals.

The called-for theoretical models should be formulated so precisely that they can be tested empirically in this society, as well as in other societies and cultures. Field studies are needed sorely in mental illness research. The replication of each study is also necessary.

I hope I have not overstressed how little we know about schizophrenia. The question is relative. If our ideal is the precise knowledge to prevent this disease, then our achievement is zero. On the other hand, when we compare what we do know with zero, our achievement is infinite. Nevertheless, the problem is still before us: what gives rise to the phenomena we call schizophrenia? The answer can come only from theoretically-oriented and methodologically-disciplined future research.

This content downloaded from 150.135.239.97 on Thu, 09 Jun 2016 17:18:06 UTC
All use subject to http://about.jstor.org/terms